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CHEMICAL MANUFACTURERS ASSOCIATION

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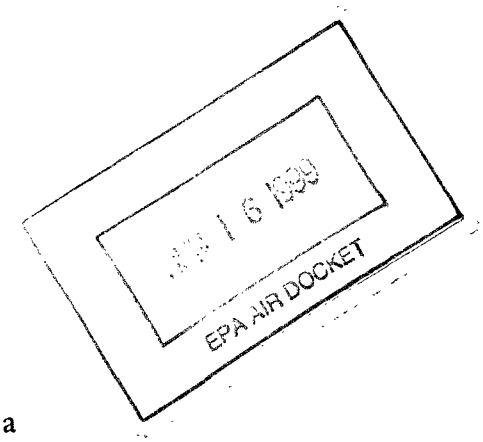
November 25, 1996



Via Certified Mail

Sally L. Shaver
Director
Air Quality Strategies and Standards Division (MD-15)
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

Re: Petition to Delist Methyl Ethyl Ketone As a HAP Under Section 112(b) of the Clean Air Act

Dear Ms. Shaver:

On behalf of the Chemical Manufacturers Association Ketones Panel, I am enclosing an original copy of a petition to remove methyl ethyl ketone (MEK) from the list of chemicals regulated as hazardous air pollutants (HAPs) under Section 112(b) of the Clean Air Act. Also enclosed is a copy of the appendices referenced in the petition.

As you know, Michael Dusetzina from your division has been designated as the contact person for purposes of processing the enclosed petition. At his request, we also are forwarding to Mr. Dusetzina two complete sets of the petition and associated appendices.

If you have any questions concerning the petition, please contact Barbara O. Francis, Manager of the Ketones Panel, at 703/741-5609.

Sincerely,

Enclosures

cc: Michael G. Dusetzina

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BEFORE THE

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

PETITION OF THE CHEMICAL MANUFACTURERS ASSOCIATION KETONES PANEL TO REMOVE METHYL ETHYL KETONE FROM THE LIST OF HAZARDOUS AIR POLLUTANTS UNDER SECTION 112(b) OF THE CLEAN AIR ACT

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EXECUTIVE SUMMARY

Pursuant to Section 112(b)(3) of the Clean Air Act (the Act), the Ketones Panel of the Chemical Manufacturers Association hereby petitions the Administrator of the Environmental Protection Agency (EPA) to remove methyl ethyl ketone (MEK) from the list of chemicals that are regulated as hazardous air pollutants (HAPs) under the Act. The Panel believes that MEK meets the statutory delisting criteria, and should therefore be removed from the list of HAPs.

Background Information

MEK is widely used as a solvent and chemical intermediate. It is a highly efficient solvent that can be used with a wide variety of resins and is therefore particularly valuable in the formulation of high-solids coatings. MEK is manufactured in a totally enclosed, continuous process that converts n-butenes into MEK.

Delisting Criteria

EPA is required to delist a substance from the HAP list if "there is adequate data on the health and environmental effects of the substance to determine that emissions, ambient concentrations, bioaccumulation or deposition of the substance may not reasonably be anticipated to cause any adverse effects to human health or adverse environmental effects." Several key aspects of this standard, as well as related statutory requirements, must be taken into account. First, as EPA has recognized, the Act does not require absolute proof that a substance will not cause any adverse effects. Rather, the Agency should use a weight-of-the-evidence approach to determine whether it is "reasonable" to anticipate that emissions of MEK will cause adverse health or environmental effects. Second, in making HAP delisting decisions, the Agency is not to consider potential accidental releases, which are regulated under Section 112(r). Rather, it must consider whether, under normal operating conditions, emissions can reasonably be anticipated to cause adverse health or environmental effects. Finally, section 112(b)(2) of the Act makes it clear that MEK should not be listed as a HAP solely because it is a volatile organic compound (VOC).

Data on Health and Environmental Effects

Acute Health Effects. MEK is not acutely toxic at the airborne concentration levels that are reasonably likely to exist beyond industrial site boundaries. Studies in laboratory animals by the oral and inhalation routes of administration show that the acute toxicity of MEK is low. In humans, exposures to between 200 and 700 ppm MEK may cause mild eye, nose and throat irritation, but are without permanent effects. No significant acute health effects are expected at exposures below these levels. Results from air dispersion modeling using EPA-approved techniques show that maximum ambient concentrations beyond industrial site boundaries are well below levels of concern for acute effects.

Chronic Health Effects. MEK also is not known to cause and cannot reasonably be anticipated to cause significant chronic health effects in humans. The EPA IRIS database recognizes that MEK "has little if any neurotoxic potential." Several studies to investigate

developmental toxicity have been conducted. The developmental NOAEL is 1,010 ppm, and the LOAEL is 3,020 ppm "based on the appearance of mild, but significant developmental effects." A 2-generation reproductive effects study has been conducted in rats in drinking water using 2-butanol, which is rapidly converted metabolically to MEK. EPA has used this study to derive an oral reference dose (RfD) for MEK of 0.6 mg/kg/day, based on decreased fetal body weights observed at 2% 2-butanol in the drinking water. The LOAEL was 3,122 mg/kg/day, and the NOAEL was 1,771 mg/kg/day. MEK was inactive in a wide variety of in vitro and in vivo genetic toxicity assays. MEK has not been tested specifically for carcinogenicity. However, the data on its structure and metabolism, the results of subchronic studies and the absence of genotoxicity indicate that MEK is not likely to have oncogenic properties.

Developing an RfC for MEK. The IRIS database includes an inhalation reference concentration (RfC) for MEK of 1.0 mg/m^3, based on the most recent developmental toxicity study. The RfC was verified in 1991, and is based on total uncertainty and modifying factors of 3,000, including a factor of 10 for interspecies extrapolation. Since that time, EPA has issued new guidance on developing RfCs. In accordance with this guidance, an uncertainty factor of 3 is used for interspecies extrapolation when dosimetric adjustments have been made, as is the case with MEK. Thus, under EPA's current methodology, the correct RfC for MEK should be 3.3 mg/m^3 (slightly greater than 1 ppm). This corrected RfC reflects MEK's low chronic toxicity, and far exceeds likely human exposures from industrial releases.

Environmental Effects. MEK also cannot reasonably be anticipated to cause adverse environmental effects. MEK has been shown to have only limited persistence in water, air and soil, and does not bioaccumulate. Studies show that MEK has a low degree of toxicity to aquatic organisms.

Data on Emissions and Exposure

Emissions Data. The Toxics Release Inventory (TRI) shows that over 2,000 facilities reported emissions of MEK in 1994. Most of these sources were very small, however, with over 85 percent reporting emissions of less than 25 tons.

Ambient Monitoring Data. MEK has been reported in ambient air at very low concentrations at a limited number of sites in rural and urban locations. Monitored levels of MEK – even in industrial areas – typically are several orders of magnitude below the RfC.

Air Dispersion Modeling Data for Industrial Facilities. The Panel funded a study by ENSR Corporation to model the maximum off-site concentrations of MEK at a wide variety of facilities emitting MEK, including the largest known sources of MEK emissions in the country. As part of this study, the Panel identified all facilities that reported MEK emissions of 200 tons or more in 1994. It contacted each of these facilities (27 based on 1994 TRI data) to gather information that could be used to model maximum off-site concentrations and also obtained additional information from public sources (including Title V permit applications). The Panel obtained the necessary data for 21 of the 27 facilities, including the 6 facilities with the highest emissions and 13 of the top 15. ENSR also developed a generic approach for modeling maximum airborne concentrations around smaller sources. The modeling data from this study shows that, for both large and small sources of MEK, the maximum annual and 24-hour airborne

concentrations beyond facility boundaries are well below levels of concern and cannot reasonably be anticipated to cause adverse health or environmental effects. The Panel also analyzed the potential that groups of sources might collectively emit significant amounts of MEK, and found that there is no such grouping.

Effect of Delisting on Emissions and Ambient Concentrations. If MEK is removed from the list of HAPs, use of MEK is likely to increase. For several reasons, however, MEK emissions are unlikely to increase substantially. MEK will continue to be regulated as a VOC and is often used in blends with other compounds that will continue to be regulated as HAPs. In addition, MEK is most widely used in paint and coating applications, where performance requirements impose inherent limits on the amount of MEK that can be used. Moreover, based on the available monitoring data and the dispersion modeling analysis conducted by ENSR, any reasonably likely increase in emissions would not be expected to result in ambient levels of concern. Perhaps most importantly, removing MEK from the HAP list is likely to decrease total VOC emissions by encouraging the use of MEK in place of other less effective solvents.

Other Considerations that Weigh in Favor of Delisting

Delisting MEK Would Help to Reduce VOC Emissions from Many Coating Operations. Over the last several years, EPA and state regulators have encouraged or required the use of high-solids coatings as an effective way to reduce VOC emissions from coating operations. It is well known that MEK is especially valuable in the formulation of high-solids coatings. The Agency for Toxic Substances and Disease Registry (ATSDR), for example, in its toxicological profile for MEK, expressly recognized that MEK "exhibits outstanding solvent properties" for a wide variety of resins. The use of MEK allows the formulation of coatings with higher solids content and lower VOC emissions. In EPA's recent rule on shipbuilding coatings, the Agency explicitly recognized that the use of highly efficient solvents such as MEK is the most effective approach for reducing VOC emissions in some coating applications. See 59 Fed. Reg. 62681, 62688 (Dec. 6, 1994).

EPA Has Recognized in Other Contexts that MEK Has Relatively Low Toxicity. In two recent rulemakings, EPA has evaluated the health effects data on MEK and concluded that MEK has relatively low toxicity. In the Agency's proposed rule under section 112(g) of the Clean Air Act, EPA developed a methodology for ranking the relative hazards of the chemicals listed as hazardous air pollutants (HAPs) and found that MEK was among the least toxic of the listed chemicals (approximately 177 out of 189). In the same rule, EPA also proposed "de minimis values" for listed HAPs. These de minimis values were intended to represent the amount of a chemical that a typical facility could emit without posing more than a "trivial" health risk. Although the de minimis values in the proposed rule were "capped" at 10 tons per year for policy reasons, the true "uncapped" de minimis value for MEK based on EPA's methodology would be 2,000 tons per year (if calculated with the current RfC in the IRIS database) or 6,600 tons per year (if the RfC is updated to reflect EPA's new RfC guidance). Even 2,000 tons is almost double the emissions of the facility reporting the highest MEK emissions in the country in 1994. EPA also evaluated the toxicity of MEK under its Significant

New Alternatives Policy (SNAP) program, and determined that it has "comparatively low toxicity."

MEK's Inclusion on the HAP List Was Not Based on a Finding of Toxicity. The initial HAP list was developed from the list of chemicals that must be reported under Section 313 of the Emergency Planning and Community Right-to-Know Act of 1986 (EPCRA). MEK was included on the Section 313 list solely because it had been included in a "Survey List" of chemicals prepared by the State of Maryland. Inclusion of MEK in the Maryland Survey List was not based on a finding of toxicity or adverse environmental effects. There is no evidence that the inclusion of MEK on the original HAP list was based on a determination by Congress, EPA or anyone else that emissions of MEK can reasonably be anticipated to cause adverse health or environmental effects.

For the foregoing reasons, and as set forth in greater detail in this Petition, the Ketones Panel respectfully urges the Administrator to remove MEK from the list of chemicals that are regulated as HAPs under the Clean Air Act.

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INTRODUCTION

Pursuant to Section 112(b)(3) of the Clean Air Act (the Act), the Ketones Panel of the Chemical Manufacturers Association (CMA) hereby petitions the Administrator of the Environmental Protection Agency to remove methyl ethyl ketone (MEK) from the list of chemicals that are regulated as "hazardous air pollutants" (HAPs) under Section 112 of the Act. The Ketones Panel includes all domestic manufacturers of MEK as well as manufacturers of several other ketone solvents.[1] MEK is a highly efficient solvent that is widely used in a variety of applications, and is particularly valuable in the formulation of high-solids paints and coatings.

Under Section 112(b)(3)(C) of the Act, EPA is required to remove a substance from the list of HAPs upon a showing that "there is adequate data on the health and environmental effects of the substance to determine that emissions, ambient concentrations, bioaccumulation or deposition of the substance may not reasonably be anticipated to cause any adverse effects to human health or adverse environmental effects." This petition reviews the considerable body of literature on the health and environmental effects of MEK, along with extensive data on releases and ambient concentrations, to show that MEK meets this standard.

In 1988, EPA reviewed the health and environmental effects data on MEK in connection with a petition submitted by the Panel asking that MEK be removed from the list of chemicals that are reportable under Section 313 of the Emergency Planning and Community Right-to-Know Act of 1986 (EPCRA). At that time, EPA identified two areas of concern,

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[1] The members of the Ketones Panel are: Eastman Chemical Company, Exxon Chemical Company, Hoechst Celanese Chemical Group, Inc., Shell Chemical Company and Union Carbide Corporation.

neurotoxicity and developmental toxicity, and the petition was withdrawn. These two concerns have now been resolved in the IRIS database based on new data for each endpoint.[2] The IRIS database recognizes that "by itself, methyl ethyl ketone has little if any neurotoxic potential." With respect to developmental toxicity, the IRIS database now recognizes a NOAEL of 1,010 ppm;[3] the fetoxicity reported at 1,126 ppm (but not at 2,618 ppm) in a 1974 study has not been replicated in two subsequent studies and has been appropriately disregarded in the IRIS database.

This Petition is presented in five parts. Part I provides general background information on MEK. Part II discusses the statutory criteria for delisting substances from the list of HAPs. Part III reviews the data on the potential health and environmental effects from exposure to MEK, and also explains how the inhalation reference concentration (RfC) for MEK in the IRIS database needs to be updated in light of EPA's 1994 guidance on setting RfCs. Part IV reviews the data that the Ketones Panel has developed on emissions and ambient concentrations of MEK, and shows that ambient MEK levels – even maximum off-site levels around the largest industrial sources of MEK emissions – are well below the updated RfC. Finally, Part V discusses several other considerations that weigh in favor of removing MEK from the HAP list. Based on the information presented in the Petition, the Panel respectfully requests that the Administrator remove MEK from the list of HAPs under Section 112 of the Clean Air Act.

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2 In light of the new data, the Panel has submitted, in addition to this Petition, a separate petition asking that MEK be delisted under Section 313 of EPCRA.

3 The IRIS RfC for MEK is based on a NOAEL of 1,010 ppm in Schwetz et al. (1991). The IRIS database also states separately that the NOAEL in that study is 1,126 ppm. This latter statement is an error; the NOAEL reported in Schwetz et al. is 1,010 ppm.

I. BACKGROUND INFORMATION

A. Chemical and Physical Properties

MEK (CAS No. 78-93-3) is a clear colorless liquid with a sharp, sweet odor and a molecular weight of 72. Common synonyms for MEK are 2-butanone and methyl acetone. It has a water solubility of 353 g/L at 10° C and a vapor pressure of 77.5 mm Hg at 20° C. The melting point and boiling point of MEK are -86.4° C and 79.6° C, respectively. The log of the octanol/water partition coefficient is 0.26 (Verschueren 1983).[4] The Henry's law constant for MEK is 1.05×10^{-5} atm-m^3/mole (Snider and Dawson 1985).

B. Production and Use

MEK is manufactured in a totally enclosed, continuous process that converts n-butenes into MEK. In the first section of the process, a mixed butenes stream (30-90$^+$% n-butenes) is contacted with a circulating sulfuric acid-water mixture. Unreacted mixed butenes are sent to the refinery for further processing. Sufficient water is added to the olefin-acid-water mixture to form secondary butyl alcohol (SBA). Some coproducts, primarily secondary butyl ether (SBE) and butene dimmer (C_8 olefins), also are formed. The SBA and coproducts are stripped from the sulfuric acid-water mixture and then separated via distillation. The water is reused in the process or treated in waste water treatment facilities. The SBA is sold or processed to MEK, the SBE is sold or used in a fuel system and the dimmer is sold.

The conversion of SBA to MEK is accomplished in a totally enclosed, continuous process. The SBA is heated to reaction temperature in a furnace and passed over a proprietary

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[4] The Table of References is found in Appendix A. Duplicate copies of all references denoted with an asterisk in the Table have been submitted with this Petition. These references are contained in Appendix K.

catalyst where it is dehydrogenated to MEK and hydrogen. Conversions and selectivity to MEK are very high. The MEK is then purified by distillation to remove water and some heavy ketones. The hydrogen is sold for its coproduct value or burned internally at plant facilities. Again, the water is either reused or treated in the waste water treatment facilities. The heavy ketones are either sold or sent to other units for further processing. The final product (99.5 wt.% MEK) is pumped to tankage and then transported to the customer via tank truck, rail car and marine loading facilities.

MEK currently is produced in the United States by three companies: Exxon Chemical Company, Hoechst Celanese, and Shell Chemical. Estimated total domestic capacity in 1995 was approximately 595 million pounds. Chemical Marketing Reporter (July 22, 1996).

MEK is used both as a solvent and as a chemical intermediate. It is a highly efficient solvent that dissolves a wide variety of resins and, therefore, is widely used in surface coatings, adhesives, inks, and traffic marking paint. As discussed in Section V.A. below, it is especially valuable in the formulation of high-solids coatings, which increasingly are being used to reduce emissions of volatile organic compounds (VOCs) from many types of coating operations. MEK also is used as a solvent in cleaning fluids and dewaxing agents, and as an extraction medium for fats, oils, waxes and resins. These and other uses of MEK are described in an Exxon technical product brochure attached as Appendix B.

C. Natural Sources of MEK

MEK is emitted to the atmosphere from such natural sources as European firs, junipers, cedars, cypress trees and ferns (Isidorov et al. 1985). In addition to being produced by various types of plants, MEK has been identified as a natural component of several foods (Lande

et al. 1976). MEK has been qualitatively identified in roasted barley, cheddar cheese, bread, honey, chicken, roasted nuts, oranges, nectarines, black tea and rum (Dumont and Adda 1978; Gordon and Morgan 1979; Takeoka et al. 1988). In addition, MEK has been detected in swiss cheese at 0.3 ppm, in cream at 0.154 to 0.177 ppm, and in milk at 0.077 to 0.079 ppm (Lande et. al. 1976), and has been detected in dried beans, split peas and lentils at 148, 110 and 50 ppm, respectively (Lovegreen et al. 1979).

II. STATUTORY CRITERIA FOR DELISTING

When Congress adopted the 1990 Amendments to the Clean Air Act, it placed 189 chemicals and chemical categories on the "initial list" of substances to be regulated as HAPs. See Section 112(b)(1). Congress recognized, however, that this initial list was not necessarily definitive, but should be reviewed and, if appropriate, revised based on the best available science. Significantly, Congress authorized the Agency not only to add to the list, but also to remove substances from the original list. It thus acknowledged the possibility that some substances on the initial list should not be regulated as HAPs.

Under Section 112(b)(3), Congress established the criteria that EPA must use in making decisions about adding or removing chemicals from the list. Under Section 112(b)(3)(C), EPA is required to remove a substance from the HAP list "upon a showing" that

> there is adequate data on the health and environmental effects of the substance to determine that emissions, ambient concentrations, bioaccumulation or deposition of the substance may not reasonably be anticipated to cause any adverse effects to human health or adverse environmental effects.

This is the basic standard under which EPA must decide whether to remove MEK from the HAP list. As discussed below, several key aspects of this standard, as well as related statutory requirements, must be taken into account.

A. Standard of Proof for Delisting

The delisting standard requires that there be "adequate" data to show that adverse effects "may not reasonably be anticipated." The Agency itself has recognized that Section 112(b) does not require absolute proof that a substance will not cause adverse effects:

> The EPA does not interpret section 112(b)(3)(C) to require absolute certainty that a pollutant will not cause adverse effects on human health or the environment before it may be deleted from the list. The use of the terms "adequate" and "reasonably" indicate that the Agency must weigh the potential uncertainties and their likely significance.

60 Fed. Reg. 48081, 48082 (Sept. 18, 1995) (proposal to remove caprolactam from the HAP list). Thus, in evaluating both the exposure data and the data on health and environmental effects, the Agency should use a weight-of-the-evidence approach to determine whether it is "reasonable" to anticipate that emissions of MEK will cause adverse health or environmental effects. The Panel believes that the data presented below clearly show that "emissions, ambient concentrations, bioaccumulation or deposition of [MEK] may not reasonably be anticipated to cause any adverse effects to human health or adverse environmental effects."

B. A Substance May Not be Listed as a HAP Unless it Reasonably Can be Expected to Cause Adverse Effects Under Normal Operating Conditions

At high exposure levels, virtually all chemicals can cause adverse health or environmental effects. Under Section 112(b)(3), however, a substance is to be listed as a HAP only if "emissions, ambient concentrations, bioaccumulation or deposition" of the substance can

"reasonably be anticipated" to result in levels that are high enough to cause such effects. Thus, if emissions of a listed substance are not reasonably expected to result in ambient levels, deposition, or bioaccumulation that reasonably can be anticipated to cause adverse health or environmental effects, then that substance meets the Section 112(b) standard for delisting. As discussed below, there is no appreciable deposition or bioaccumulation of MEK, and ambient concentrations are far below levels that reasonably may be expected to cause adverse effects.

In this regard, it is significant that accidental chemical releases are addressed in another part of the Act, Section 112(r). Section 112(b)(2) specifically states that accidental releases that are subject to regulation under Section 112(r) are not to be considered in HAP listing decisions. Thus, it is clear that listing and delisting decisions must be made based on exposure levels that result from normal or routine emissions, not from accidental releases.

C. MEK's Status as a VOC Is Not Relevant to the Decision of Whether it Should be Listed as a HAP

Like most solvents, MEK is a volatile organic compound (VOC). VOCs are regulated as ozone precursors under Title I of the Act because they can react photochemically with other pollutants to form ground-level ozone. Congress made it clear, however, that a substance is not to be listed as a HAP solely because it is a VOC. Section 112(b)(2) of the Act provides that a substance which is a precursor to a pollutant (such as ozone) that is listed under Section 108(a) of the Act may not be included on the HAP list unless it "independently meets" the HAP listing criteria. The listing criteria under Section 112 are focused on direct toxic effects, not on secondary effects that may result from the formation of ozone. Substances that meet the HAP listing criteria include those which "are known to be, or may reasonably be anticipated to be, carcinogenic, mutagenic, teratogenic, neurotoxic, which cause reproductive dysfunction, or

which are acutely or chronically toxic." Section 112(b)(2). Thus, the fact that a substance may be an ozone precursor is not relevant to the decision of whether it should be listed as a HAP. The Agency implicitly recognized this fact by removing caprolactam, which is a VOC, from the list of HAPs. See 61 Fed. Reg. 30,816 (June 18, 1996).

As a practical matter, it is also unnecessary to use Section 112 of the Clean Air Act to regulate VOC emissions. There are many other programs under the Clean Air Act that are specifically designed to control emissions of VOCs and other ozone precursors. Under Section 110 and Part D of the Act, any state that does not meet the national ambient air quality standard (NAAQS) for ozone must adopt a state implementation plan to regulate VOC emissions from both new and existing sources. In addition, VOC emissions are regulated under Section 111 (new source performance standards) and Part C (prevention of significant deterioration). In light of the other programs designed specifically to control VOC emissions, it is not surprising that Congress decided that VOCs should not be regulated as HAPs unless they "independently meet" the listing criteria under Section 112.

III. DATA ON HEALTH AND ENVIRONMENTAL EFFECTS

There is a substantial body of toxicological literature on MEK. This section of the Petition first reviews the potential exposure pathways and explains why inhalation is the only significant route of human exposure potentially resulting from MEK emissions. The Petition then reviews the available literature on the health and environmental effects of MEK, and demonstrates that MEK cannot reasonably be anticipated to cause adverse health effects or adverse environmental effects. The Petition also discusses the RfC in the IRIS database and

explains why it needs to be adjusted from 1.0 mg/m^3 to 3.3 mg/m^3 to be consistent with EPA's current guidance for setting RfCs.

A. Inhalation Is the Only Significant Route of Human Exposure to MEK Emissions

Section 112(b)(2) indicates that, in making listing decisions, the Agency should consider whether a substance may reasonably be anticipated to cause adverse effects "through inhalation or other routes of exposure." In light of the reasonably anticipated ambient concentrations (described in Section IV of the Petition), it is clear that humans would not be expected to ingest any appreciable amounts of MEK resulting from air emissions. Further, because of MEK's relatively ready biodegradation and rapid volatilization in water (see Section III.D), it is highly unlikely that humans will be exposed to significant amounts of MEK in drinking water. In addition, given its lack of persistence and low bioaccumulation potential (also described in Section III.D), MEK emitted to the air would be unlikely to concentrate in food sources. Finally, dermal absorption is likely to be insignificant compared to inhalation, both because dermal absorption is a less efficient exposure route to humans and ambient concentrations of MEK are not high enough to make this route toxicologically relevant. Thus, it is clear that inhalation is the only route of human exposure with potential significance.

B. MEK Cannot Reasonably be Anticipated to Cause Adverse Acute Health Effects In Humans

The available data show that MEK's acute toxicity is low. MEK cannot reasonably be anticipated to cause acute health effects in humans at concentration levels that are likely to exist beyond facility boundaries.

MEK's acute toxicity in animals has been shown to be very low. Estimates of the acute oral LD_{50} in rats range from approximately 2.5 to 5.6 g/kg (Kimura et al. 1971; Smyth et al. 1962). An oral LD_{50} in mice of 4.05 g/kg was reported by Tanii et al. (1986). Estimates of the acute dermal LD_{50} for rabbits range from greater than 5.0 g/kg (Opdyke 1977) to 8.0 g/kg (Smyth et al. 1962). There have been several attempts to estimate the acute inhalation LC_{50} in guinea pigs. In the first of these studies (Patty et al. 1935), guinea pigs tolerated 10,000 ppm for up to 13 hours, and no effects were seen at 3,300 ppm. In a second guinea pig study, the 4 hour inhalation LC_{50} was found to be between 10,000 and 33,000 ppm (Specht et al. 1940).

Two inhalation studies in rats indicate 8-hour LC_{50} values of approximately 8,000 ppm (Pozzani et al. 1959; Smyth et al. 1962). A somewhat higher 4-hour LC_{50} of 11,700 ppm was reported by LaBelle and Brieger (1955). In a subsequent study (Carpenter et al. 1949), the two-hour LC_{50} was estimated to be between 2,000 and 4,000 ppm. It is likely, however, that the lower value reported in the Carpenter study was an error. A series of more recent studies evaluated the subchronic toxicity and/or neurotoxic potential of MEK. In one of those studies, rats were exposed to MEK at 1250, 2500 or 5000 ppm for 6 hours/day, 5 days/week for 90 days. These levels of exposure were minimally toxic (Cavender et al. 1983). In a second study, exposure to 10,000 ppm was not lethal over a period of "a few" (actual number unspecified) days (Altenkirch et al. 1978). Thus, the best estimate is that the 4 hour inhalation LC_{50} in rats exceeds 5,000 ppm and may exceed 10,000 ppm.

Several studies have been conducted to evaluate the potential acute toxicity of MEK to humans. In some cases humans were exposed under laboratory conditions, whereas other studies involved assessments of effects of occupational exposure. In a study by Nelson et

al. (1943), human volunteers were exposed for three to five minutes to differing vapor concentrations. MEK reportedly produced "slight" nose and throat irritation at 100 ppm in an unspecified number of subjects. Mild eye irritation was reported by some subjects at 200 ppm, but the majority of test subjects concluded that 200 ppm MEK would be tolerable for an 8-hour work day. Exposure to 300 ppm was considered objectionable. Based on these observations, the authors recommended a maximum occupational exposure level of 200 ppm.[5]

Elkins (1959) has reported an investigation of industrial exposure to MEK prior to 1950. In certain processes in Massachusetts, exposures reportedly were as high as 700 ppm. Concentrations above 300 ppm reportedly resulted in complaints of headaches and throat irritation, and, in one plant, nausea and vomiting were reported with concentrations reportedly averaging 500 ppm. However, no permanent effects were noted.[6]

More recently, Dick et al. (1984, 1988, 1989, 1992) tested the neurobehavioral potential of exposure to 200 ppm MEK for 4 hours in humans.[7] Over 100 men and women were examined in this study and a large number of neurobehavioral parameters were reviewed. The study also evaluated the following "subjective effects": odor presence, strong odor,

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5 The reports of "slight" and "mild" irritation in the Nelson study should be interpreted with caution as they likely overstate MEK's potential to cause irritation. The use of naive subjects and the short duration of exposure make it difficult to determine whether the subjective responses reflected odor perception or true sensory irritation. It is well-known that odor can influence subjective responses to chemical exposure (Cavalini et al. 1991; Kasko et al. 1990).

6 The study of Elkins also needs to be interpreted with caution because of the reliance on subjective complaints and incomplete information about peak exposures, exposures to other compounds, and other aspects of the work conditions.

7 This series of studies is discussed further in Section III.C.2 (Neurotoxicity).

objectionable odor, headache, nausea, throat irritation, tearing and unpleasant odor. The results showed no statistically significant increase in any subjective effects between subjects exposed to 200 ppm MEK for 4 hours and controls, except for a finding of "strong odor." Dick et al. (1992).

OSHA has established a permissible exposure limit (8-hour TWA) for MEK of 200 ppm. ACGIH recommends an 8-hour threshold limit value (TLV) of 200 ppm and a 15 minute short term exposure limit (STEL) of 300 ppm.

In summary, human experience reveals that acute exposures to MEK at levels above 200 ppm may cause mild eye, nose, and throat irritation, but produce no permanent adverse health effects. As explained in Section IV of this Petition, actual ambient concentration levels, including off-site concentrations at the largest industrial emitters of MEK, are expected to be significantly below this level. Maximum annual average off-site concentrations are typically well below 1 ppm, and maximum 24-hour average off-site concentrations are typically below 3 ppm, based on EPA-approved air dispersion modeling techniques. Thus, the available evidence shows that MEK should not be listed as a HAP based on concerns about potential acute health effects.

C. MEK Cannot Reasonably be Anticipated to Cause Adverse Chronic Health Effects In Humans

There is a considerable body of data which shows that MEK cannot reasonably be anticipated to cause chronic health effects in humans at reasonably anticipated ambient levels. In this regard, the Panel notes that EPA's Health and Environmental Review Division (HERD) reviewed the toxicological database on MEK in 1988 in connection with a petition submitted by the Panel to seek delisting of MEK under Section 313 of the Emergency Planning and Community Right-to-Know Act of 1986 (EPCRA). At that time, EPA identified two areas of

concern, neurotoxicity and developmental toxicity, and the petition was withdrawn. These two concerns have now been resolved in the IRIS database based on new data for each endpoint. With respect to neurotoxicity, the IRIS database now states that "by itself, methyl ethyl ketone has little if any neurotoxic potential." With respect to developmental toxicity, the IRIS database now recognizes a NOAEL of 1010 ppm based on Schwetz et al. (1991). These two issues, as well as the other chronic health effects data, are discussed in more detail below.[8]

1. **Subchronic Studies**

Subchronic inhalation studies show that MEK has little or no subchronic toxicity. Male and female Fischer 344 rats were exposed to 0, 1250, 2500, or 5000 ppm MEK vapors 6 hours/day, 5 days/week for 90 days (Cavender et al. 1983). This treatment produced no evidence of adverse effects on the clinical health or growth of either sex except for a depression of mean body weight in the 5,000 ppm exposure group. The 5,000 ppm animals had a slight but significant increase in liver weight, liver weight/body weight ratio, and liver weight/brain weight ratio. Serum glutamic pyruvic transaminase (SGPT) activity was elevated in female rats from the 2,500 ppm exposure group, but was significantly reduced in the 5,000 ppm group. In addition, alkaline phosphatates, potassium and glucose values for female rats in the 5,000 exposure group were increased.

2. **Neurotoxicity**

Numerous studies have been conducted to assess the neurotoxic potential of MEK. None of the studies provides any evidence that MEK produces nervous system damage.

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[8] As noted in the introduction to this Petition, in light of this new data, the Panel also has submitted a new petition seeking the delisting of MEK under Section 313 of EPCRA.

The IRIS database concluded that "by itself, methyl ethyl ketone has little if any neurotoxic potential." The IRIS database states further, "at present, there is no convincing experimental evidence that methyl ethyl ketone, by itself, is neurotoxic to either experimental animals or humans other than possibly inducing CNS depression at high exposure levels."

In the series of human studies by Dick et al. (1984, 1988, 1989, 1992) (described in Section III.B, supra), no significant differences between the exposed and control populations were observed for any of the neurobehavioral parameters. The studies included five psychomotor tests: choice reaction time, simple reaction time, visual vigilance, dual task (auditory tone discrimination and tracking), and memory scanning. One sensorimotor test (postural sway) also was included. The IRIS database states, "No statistically significant changes in neurobehavioral performance were observed."

Saida et al. (1976) evaluated the effects of MEK alone and in combination with methyl-n-butyl ketone (MnBK) in Sprague-Dawley rats. Animals were exposed continuously (i.e., 24 hours/day) to MEK at 1,125 ppm; to a mixture of MnBK and MEK that consisted of MnBK at 225 ppm and MEK at 1,125 ppm; or to MnBK at 225 ppm. Rats were sacrificed 16, 25, 35, and 55 days after initiation of treatment. Nerve tissue was collected for microscopic examination, and quantitative histological studies were performed. No peripheral neurotoxicity was seen with MEK alone. In addition, it was reported that further studies were carried out for as long as 5 months without evidence of abnormality. However, the authors did report that the combination of MnBK and MEK was more toxic than MnBK alone as measured by onset to clinical paralysis and by several histological parameters.

A study by Spencer et al. (1976) evaluated the neurotoxic potential of MEK alone and in combination with MnBK and methyl isobutyl ketone (MIBK) in cats. Test materials were administered by subcutaneous injection twice daily, 5 days per week for up to 8.5 months. Selected tissues were removed and examined by light and electron microscopy. Chronic intoxication with commercial grade MEK alone produced no clinical or pathological evidence of neuropathy. Animals treated with a 9:1 mixture of commercial grades of MEK and MnBK failed to develop clinical neuropathy, although pathological evidence of nerve damage was present. The subclinical damage appeared to be in proportion to the amount of MnBK used, although enhancement by MEK of the neurotoxic effects of MnBK could not be excluded.

A study by Altenkirch et al. (1978) evaluated the neurotoxic potential of MEK alone and in combination with n-hexane. Rats were exposed 8 hours/day, 7 days/week for 15 weeks. MEK was initially present at 10,000 ppm, but this level was reduced to 6,000 ppm after several days because of severe irritation to the upper respiratory tract. As in the other studies, the animals were sacrificed and perfused, and sections of nerve tissue were examined microscopically. Rats exposed to MEK did not develop any obvious motor impairment up to the seventh week when all animals died without neurological symptoms. Histopathological examinations revealed severe signs of bronchopneumonia in all animals. There was no evidence of histological alterations in the nerve tissue. Thus, MEK was found to be without neurotoxic potential. However, there was some evidence that MEK potentiated the neurotoxic effects of n-hexane.

The study by Cavender et al. (1983), described in III.C.1 (subchronic studies), provides further evidence that repeated inhalation exposures (at doses up to 5,000 ppm) produce

no clinical or pathological evidence of neurotoxic effects in rats. No histophathological lesions were reported in the brain, sciatic nerve, tibial nerve, spinal cord, or optic nerves, nor were any effects reported in posture, gait, tone, or symmetry of the facial muscles, or in the pupillary, palpebral, extensor thrust, and cross-extensor thrust reflexes. A slight but statistically significant increase in brain weight was reported in female rats exposed to 5,000 ppm, but no pathological changes were reported in the medulla oblongata or the sciatic and tibial nerves. Based on the Cavender study, ECETOC recently concluded that the neurotoxicity NOAEL in rats for MEK is 5,000 ppm (ECETOC 1996). Thus, the earlier observation by Saida et al. (1976) that MEK was not neurotoxic in rats, even following repeated exposure at relatively high levels, was confirmed.

The effect of MEK exposure on neurophysiology also was evaluated by Takeuchi et al. (1983). Rats were exposed 12 hours a day for 24 weeks to 200 ppm MEK vapor. The rats showed significant increases in motor nerve conduction velocity and mixed nerve conduction velocity and reduced distal motor latency after four weeks of exposure. However, significant effects were not observed at any other times. The biological significance of these findings is uncertain. The effects were transitory in nature, and in other studies there was no evidence of behavioral changes or microscopic damage to nerve tissue at much higher exposure levels or following exposure for longer periods of time. The IRIS database notes that the alteration in nerve conduction velocity at four weeks "was not corroborated by histopathology and was in the opposite direction to that predicted by peripheral neurotoxins such as n-hexane."

In summary, the above studies show that MEK has essentially no neurotoxic potential. The available studies demonstrate further that MEK does not pose a neurotoxicity hazard to humans under realistic exposure scenarios.

3. Genotoxicity

MEK has been shown to be without genotoxic activity in a variety of short term tests. Among the tests which produced negative results are assays for point mutation (e.g., Salmonella, E. coli, Saccharomyces cerevisiae and mouse lymphoma), chromosomal aberration (rat liver cells in vitro and mouse bone marrow in vivo), DNA damage (unscheduled DNA synthesis in rat hepatocytes), and morphologic transformation (BALB 3T3 morphologic transformation assay) (NTP Fiscal Year 1987 Annual Plan; Florin et al. 1980; Marnett et al. 1985; Nestmann et al. 1980; Perocco et al. 1983; Brooks et al. 1988; O'Donoghue et al. 1988; Smirasu 1976). There was some evidence that MEK induced aneuploidy in yeast (Zimmerman et al. 1985), but this does not appear to be biologically important since negative results were obtained in a mouse micronucleus test (O'Donoghue et al. 1988).

4. Developmental Toxicity

Several studies have investigated the effects of MEK on fetal development following inhalation exposure. In the most recent study, pregnant Swiss mice were exposed to 0, 398, 1010, or 3020 ppm MEK for 7 hours/day on gestation days 6-15 (Schwetz et al. 1991; Mast et al. 1989). The only maternal effect observed was a concentration-related increase in relative liver and kidney weight, which was statistically significant in the 3,020 ppm dams. A decrease in fetal body weight also was seen at this concentration; however, it was statistically significant only in the male fetuses. Also in the 3,020 ppm group, there was a statistically significant increase in the incidence of misaligned sternebrae when measured on a fetus, but not litter, basis. No significant increase in any single malformation was seen. Neither maternal nor developmental toxicity was observed at or less than 1,010 ppm. EPA has identified 1,010 ppm

as the NOAEL, and 3,020 ppm at the LOAEL, based on mild, but significant developmental effects (IRIS 1991).[9]

In the earliest developmental toxicity study (Schwetz et al. 1974), pregnant Sprague-Dawley rats were exposed to 0, 1126, or 2618 ppm MEK for 7 hours/day on days 6-15 of gestation. No maternal effects or effects on fetal resorption were seen. At the 1,126 ppm dose level, there was a decrease in fetal body weight and crown-rump length, but this was not observed in the 2,618 ppm rats. There were no significant increases in gross, soft tissue, or skeletal effects in litters of dams exposed to 1,126 ppm MEK. In the 2,618 ppm group, there was a significant increase in the number of fetuses and litters with gross anomalies. No statistically significant specific soft tissue malformation or alteration was seen; however, the total number of litters with abnormal fetuses was significantly greater than controls.

In an attempt to confirm the effects reported by Schwetz et al. (1974), another study was conducted in rats (Deacon et al. 1981). In this study, groups of pregnant Sprague-Dawley rats were exposed to 0, 412, 1002, or 3004 ppm MEK for 7 hours/day on gestation days 6-15. Decreased maternal body weight and increased water consumption was seen in the 3,004 ppm group; however, no other maternal effects were noted. No statistically significant differences were seen in the 412 and 1,002 ppm groups. In the 3,004 ppm group, there was no difference in malformation frequency, but extra ribs and delayed ossification were seen. There were no differences in the number of fetuses/litter, fetal body weight, or crown-rump length at

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[9] EPA has used this study to calculate an RfC for MEK. See discussion in Section III.C.8 (pp. 24-25).

the highest dose level tested. Thus, the study by Deacon et al. did not confirm the effects observed at the mid-dose only in the earlier study by Schwetz et al.

A fourth developmental toxicity study has been conducted with 2-butanol. (Nelson et al. 1989). Data on 2-butanol are relevant because it has been experimentally determined that approximately 96% of an administered dose of 2-butanol is metabolically converted to MEK (Traiger and Bruckner 1976). (As described in the next section, a 2-generation reproductive effects study of 2-butanol has been used by EPA to determine the oral RfD for MEK in IRIS.) In the 2-butanol study, pregnant Sprague-Dawley rats were exposed by inhalation to 0, 3500, 5000, or 7000 ppm 2-butanol for 7 hours/day on gestation days 1-19. At concentrations of 5,000 and 7,000 ppm, the maternal rats exhibited narcosis and impaired gait during the exposure period, and the 7,000 ppm group did not fully recover by the next exposure day. Maternal body weight gain and food consumption were reduced at all exposure levels. Statistically increased incidences in resorptions/litter and decreases in live fetuses/litter were seen in the 7,000 ppm group. There were also significant reductions in fetal body weight at 5,000 and 7,000 ppm. This study demonstrates that 2-butanol is not teratogenic and is not developmentally toxic except at very high concentrations that produce significant maternal effects.

The 2-generation reproductive effects study on Wistar rats (described in the next section) also included a teratologic phase in which the parent dams were rebred (2nd litters) and subjected to cesarean section on day 20 of gestation after being exposed to 2-butanol at 0, 0.3, 1.0 and 2% in drinking water (Cox et al. 1975). Pregnancy rates and survival of these females were unaffected. The body weight of the pregnant dams was not depressed. Examination of

uterine contents on the 20th day of gestation suggested that 2-butanol was somewhat fetotoxic at the 2% dosage level, as shown by the decreased mean pup weights. This response was minimal, however, as indicated by the lack of any effect on implantation or the occurrence of early or late fetal deaths. There were no significant soft tissue findings in the group treated at the 2% dosage level.

In summary, the developmental toxicity potential of MEK (or 2-butanol, a metabolic surrogate) has been studied in rats and mice by inhalation and in rats following oral exposure. In all these studies, MEK produces some developmental toxicity, but not teratogenicity, at the highest dose examined. In most of these studies, the high dose effects were associated with maternal toxicity. EPA has identified a developmental NOAEL of 1,010 ppm and a LOAEL of 3,020 ppm based on mild effects. There is no evidence to suggest that MEK poses a developmental toxicity hazard at concentrations likely to be present in the environment from realistic exposure scenarios.

5. Reproductive Toxicity

No classic reproductive effects studies have been conducted with MEK. However, results of subchronic inhalation studies do not indicate that the reproductive organs of either sex are likely target organs for MEK. In the study by Cavender et al. (1983), histological examination of the testes, epididymides, seminal vesicles, vagina, cervix, uterus, oviducts, ovaries, and mammary glands of rats exposed to MEK at concentrations up to 5,000 ppm for 90 days revealed no exposure-related lesions.

Further, a 2-generation reproductive effects study has been conducted with 2-butanol. As described in the preceding section, 2-butanol is rapidly converted to MEK, such that

2-butanol studies are useful for evaluating the potential toxicity of MEK. In the 2-generation study, male and female Wistar rats were exposed to 2-butanol at 0, 0.3, 1.0 or 3.0% in drinking water (Cox et al. 1975). After 9 weeks of exposure, the parental animals were mated. Significant effects were noted in the F_{1A} litters from the 3% group, including a reduced number of live pups, pup viability, and mean body weights at 4 and 21 days. Based on these effects, the 3% butanol dose was decreased to 2%. Following a 2 week adaptation period, the P generation subsequently was remated to produce a second litter (F_{1B}) and the F_{1A} animals were selected for an F_2 mating. Similar effects were seen in the F_{1B} pups as in the F_{1A} pups, though the severity was reduced. As reported in the IRIS database, the critical effect seen in these studies was decreased fetal body weight. Based on this critical effect, a LOAEL of approximately 3,122 mg/kg/day (2% solution) and a NOAEL of 1,771 mg/kg/day (1% solution) were identified. Based on this NOAEL, the EPA has calculated an oral RfD for MEK of 0.6 mg/kg/day.

6. Carcinogenicity

MEK has not been tested specifically for carcinogenicity because data on its structure and metabolism, subchronic health effects, and genotoxicity indicate that MEK is not likely to have carcinogenic properties. MEK does not belong to a class of chemicals known to react with DNA, nor is it metabolized to materials that are likely to react with DNA. Materials which are oncogenic for mammals appear to cause cancer either by interacting with the genetic material (DNA) (that is, they are genotoxic and, therefore, are probably initiators of the carcinogenic process), or they produce chronic toxic effects which result in increased cell turnover and, therefore, produce effects by epigenetic mechanisms and are probably promoters of the carcinogenic process. The data available for MEK indicate that this chemical substance is

not genotoxic and also does not produce significant cumulative toxicity. Therefore, MEK is unlikely to be carcinogenic by either genetic or epigenetic mechanisms and is unlikely to be either an inducer or promoter of carcinogenicity.

Although MEK has not been tested specifically for carcinogenicity, it has been used as a solvent for the investigation of the contribution of elemental sulfur and other organic sulfur compounds to dermal carcinogenesis in C3H male mice (Horton et al. 1965). No skin tumors were induced as a result of applying 50 mg of a 17% MEK solution to each mouse topically twice a week for one year.

Two studies have been conducted in workers exposed to MEK in dewaxing plants. Although the studies involved relatively small populations exposed to relatively low levels of MEK, each study reported that deaths due to cancer were less than expected. In the first study, no overall excess in cancer incidence was found in a cohort of 446 males; 13 cancer deaths were observed compared to 14.26 expected. (Alderson and Rattan 1980). An increase in cancer of the buccal cavity and pharynx was observed, based on very small numbers (2 observed compared to 0.13 expected). The overall cancer incidence also was less than expected in a second study of 1,008 male oil refinery workers exposed to 1-4 ppm MEK in a dewaxing-lubricating oil plant. (Wen et al. 1985). No increase in buccal and pharyngeal cancers was observed in this second study. ATSDR reviewed these studies and concluded that "preliminary epidemiological studies suggest that occupational exposure to [MEK] does not increase the development of neoplasms." (ATSDR 1992 at p. 49).

7. Other Effects

The study by Cavender et al. (1983), described in Section III.C.1 (subchronic studies), also evaluated the findings of pulmonary irritation and the broncho-pneumonia reported by Altenkirch et al. (1978). There was no evidence of upper respiratory irritation in the Cavender study at doses up to 5,000 ppm.

Although MEK is not neurotoxic, it has been shown to potentiate the effects of MnBK (Saida et al. 1976), n-hexane (Altenkirch et al. 1978), and 2,5-hexanedione (Ralston et al. 1985). This effect appears to be due to the persistence of 2,5-hexanedione (the neurotoxic metabolite of n-hexane and MnBK) in the blood. Although the mechanism has not been firmly established, the data support the hypothesis that MEK may be a competitive inhibitor for 2,5-hexanedione metabolism. The neurotoxic effects of 2,5-hexanedione and its parent compounds ("the gamma diketones") have been well studied, and the environmental releases of such compounds are likely to be well below levels that might pose a concern based on potential potentiation by MEK.

Several authors have studied the interaction between MEK and chemicals which cause hepatotoxicity and reduced bile flow (e.g., Dietz and Traiger 1979; Hewitt et al. 1986; Hewitt et al. 1983; Traiger et al. 1975). In one of these studies (Dietz and Traiger 1979), pretreatment with 2.1 ml/kg MEK enhanced the hepatotoxic response to 0.1 ml/kg carbon tetrachloride. It has been hypothesized that this effect is related to the stimulatory effect of MEK on the drug metabolizing system of the endoplasmic reticulum (Traiger et al. 1975). Subsequent studies demonstrated that pretreatment with MEK (15 mmol/kg) also potentiated the hepatotoxic potential of chloroform (Hewitt et al. 1983). A subsequent mechanistic study established that the

potentiating effects of MEK were not due to the induction of cholestasis (Hewitt et al. 1986). Thus, it is likely that the potentiating activity of MEK is related to effects on mixed function oxidase activity in the liver.

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Overall, the weight of the available evidence shows that MEK cannot reasonably be anticipated to cause chronic health effects in humans. The IRIS database recognizes that MEK "has little if any neurotoxic potential." Four developmental toxicity studies (one using 2-butanol) show that MEK causes only mild effects at high concentrations. Similarly, the 2-generation reproductive effects study using 2-butanol showed only mild effects at high oral doses. MEK has been shown to be inactive in a wide variety of in vitro and in vivo genetic toxicity assays, and there is no evidence to suggest that MEK poses a cancer hazard to animals or humans. Further, the relatively low toxicity of MEK is reflected in the IRIS oral RfD of 0.6 mg/kg/day and the updated inhalation RfC of 3.3 mg/m^3 (described further below).

8. Using Current EPA Methodology, the RfC for MEK Is 3.3 mg/m^3

EPA has identified Schwetz et al. (1991) as the appropriate study to use as the basis for setting an RfC for MEK (IRIS 1991). As noted above, the Agency identified 1,010 ppm as the NOAEL, and 3,020 ppm at the LOAEL, based on mild, but significant developmental effects. Based on the NOAEL, EPA applied safety and uncertainty factors totaling 3,000 to derive an RfC of 1.0 mg/m^3. Included in the uncertainty factors was a factor of 10 for interspecies extrapolation.

The RfC was verified in the IRIS database in 1991. Since that time, EPA has published new guidance for deriving RfCs. See EPA Office of Research and Development,

"Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry," EPA No. 600/8-90/066F (October 1994) (hereinafter the "1994 RfC Guidance"). The new guidance states that, if standard default dosimetric adjustments have been made, an uncertainty factor (UF) of 3 should be used for interspecies extrapolation rather than an UF of 10.[10] Id. at 4-76 to 4-78. Since the 1994 RfC Guidance was issued, the Agency has used this approach for setting a number of RfCs in the IRIS database.

The IRIS database clearly indicates that default dosimetric adjustments were made in the case of MEK. IRIS Database Entry for MEK (attached as Appendix C). However, because the RfC was established before the 1994 RfC Guidance was adopted, a UF of 10 for interspecies extrapolation, rather than a UF of 3, was used to derive the current RfC. Using the UF of 3 for interspecies extrapolation reduces the total uncertainty factor from 3,000 to 900, and produces a corrected RfC value of 3.3 mg/m^3 (slightly greater than 1 ppm).[11]

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[10] The new guidance also states that, "[i]f more rigorous [dosimetric] adjustments can be made, an additional reduction of the UF would be warranted." 1994 RfC Guidance at 4-78.

[11] This corrected RfC should be considered a conservative value because it is designed to allow continuous exposure for a lifetime of 70 years without adverse effect. Moreover, in the case of MEK, an UF of 10 for incomplete database probably is excessive because a 2-generation reproductive effects study using 2-butanol has been conducted (see Section III.C.5 of the Petition), and experience with other compounds shows that an UF of 10 for lack of a chronic study usually is higher than necessary (Dourson and Stara 1983). The necessity of the modifying factor of 3 also is open to question.

D. MEK Does Not Cause Significant Adverse Environmental Effects

Under Section 112(b)(3)(C), EPA must also consider whether emissions of a substance may reasonably be anticipated to cause "adverse environmental effects." The term "adverse environmental effect" is defined as:

> any significant and widespread adverse effect, which may reasonably be anticipated, to wildlife, aquatic life, or other natural resources, including adverse impacts on populations of endangered or threatened species or significant degradation of environmental quality over broad areas.

Section 112(a)(7). Thus, to qualify as an "adverse environmental effect" for purposes of delisting decisions, the effect must be both "significant and widespread." As discussed below, MEK emissions cannot reasonably be anticipated to cause significant or widespread adverse effects on the environment.

1. Persistence and Bioaccumulation

MEK has limited persistence in water, soil and air, and low bioaccumulation potential because of its physical and biological properties. See EPA memorandum from J.V. Nabholz to E. Dage, Chemical Review and Evaluation Branch at pp.1-2 (November 2, 1988) (MEK "has low bioconcentration potential; rapidly evaporates from water and soil; moderately biodegrades under aerobic conditions in aquatic environments . . . is more persistent in aerobic soils, but not very much more.") (hereinafter "Nabholz Memorandum," which is attached as Appendix D).

Studies show relatively rapid degradation and/or evaporation of MEK in all three environments. The primary route of loss from terrestrial and aquatic ecosystems will be through volatilization to the air. MEK is expected to biodegrade relatively rapidly in aerobic

environments and, under anaerobic conditions, biodegradation will also occur after a period of acclimation. Because MEK is not expected to persist and because it has a low calculated log K_{OW} (octanol/ water partition coefficient), low log K_{OC} (adsorption coefficient), and low BCF (bioconcentration factor), MEK should not bioaccumulate in the environment. See Nabholz Memorandum at p. 2.

MEK released to terrestrial environments has the potential to leach into the soil, but because of its high vapor pressure, 90.6 mm Hg at 25° C (Ambrose et al. 1975), under most conditions it would largely volatilize to the air. Henry's Law constant for MEK, 1.05×10^{-5} atm-m^3 /mole (Snider and Dawson 1985), indicates that volatilization from water will occur at a significant rate (Lyman et al. 1982). MEK in aquatic environments is expected to evaporate to the air with estimated half-lives of approximately 3 and 12 days in rivers and lakes, respectively (Howard et al. 1990).

MEK released into the atmosphere will degrade principally by reacting with photochemically produced hydroxyl radicals. The half-life for MEK from hydroxyl radical attack is 2.3 days (Cox et al. 1981). MEK also can absorb radiation and is subject to photolysis, resulting in additional degradation.

Results of standardized aerobic biodegradation testing using a sewage inoculum in fresh water showed that MEK biodegraded to 76 and 89 percent in 5 and 20 days, respectively, and to 32 and 69 percent in salt water for the same respective incubation periods (Price et al. 1974). Additional testing using an inoculum of filtered effluent from a wastewater treatment plant showed that MEK biodegraded to 83 percent in 5 days (Bridie et al. 1979), which confirmed the rapid biodegradability seen in previous test results. Anaerobic biodegradation of

MEK also was demonstrated to occur after a one-week acclimation period (Chou et al. 1979), but this is expected to occur at a slower rate as compared with biodegradation under aerobic conditions.

Partitioning of MEK to sediment or biota most likely will not be significant, based on a calculated log K_{OW} of 0.26 and log K_{OC} of 1.48 (ASTER 1992). These values suggest that MEK would have a low potential to sorb and therefore accumulate in sediment. See Nabholz Memorandum at p. 2 (MEK "is very water soluble and does not sorb to soil particles"). MEK also has a calculated fish bioconcentration factor of 1.0 (ASTER 1992), and thus would not be expected to bioconcentrate in aquatic species.

2. Environmental Effects

Standardized toxicity testing has demonstrated that MEK has very low toxicity to aquatic organisms (see Appendix E for a summary of aquatic toxicity data). Although there are no data on chronic aquatic toxicity, MEK is not expected to be chronically toxic to aquatic organisms because it has limited persistence in aqueous habitats due to volatilization, and biotic and abiotic degradative processes are expected to contribute to its rapid removal from aquatic habitats. Acute studies in two freshwater fish, fathead minnow (Brooke et al. 1984) and bluegill sunfish (Turnbull et al. 1954), indicate LC_{50} concentrations of 3,220 mg/L (96 hours) and 5,640 mg/L (48 hours), respectively.[12] The freshwater invertebrate, *Daphnia magna*, was shown by Randall and Knopp (1980) to have a 48-hour EC_{50}IM concentration of 5,091 mg/L.[13]

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[12] The LC_{50} is the concentration at which 50% of the test population does not survive.

[13] The EC_{50}IM is the concentration at which 50% of the test population is immobilized.

Heitmuller et al. (1981) demonstrated that a salt water fish, sheepshead minnow, did not show any adverse toxic effects to MEK after 96 hours at a concentration of 400 mg/L. Since this was the highest concentration tested, the actual no effect concentration is most likely greater than this level. Price et al. (1974) measured the 24-hour LC_{50} concentration as 1,950 mg/L for the marine invertebrate, *Artemia salina.*

3. Environmental Monitoring

Concentrations of MEK in the environment (air, water, soil), as determined by various monitoring studies, have been shown to be low to non-detectable in areas where MEK is manufactured, transported and used. See, e.g., Houston Regional Monitoring Study (Ongoing), Bozelli, et al. (1980) and Baton Rouge Study (1988), which are summarized in Section IV.B (data on ambient air concentration levels).

The EPA found MEK in only one aqueous sample in a 1977 environmental sampling program covering 204 sites (Ewing et al. 1977). A concentration of 23 ppb was detected at a sampling station in the Burbank Western Wash which flows into the Los Angeles River in California. MEK was not detected at the next station downstream at South Gate, California.

In an industrial effluent study, Jungclaus et al. (1978) measured MEK concentrations in wastewater, river water and sediments. Although MEK was found at concentrations ranging from 8.0 to 20.0 ppm in wastewater, it was not detected in receiving river water and sediment.

Corwin (1969) measured volatile organics, including MEK, in marine environments. He reported MEK concentrations in samples taken from the Straits of Florida in

the United States and from the eastern Mediterranean Sea. All values reported were less than 22 ug/L. Corwin suggested that natural sources of ketones, such as volcanoes, forest fires, biodegradation intermediates, and natural production by organisms, were possible sources contributing to MEK found in marine environments.

* * *

In summary, MEK cannot reasonably be anticipated to cause a significant adverse effect on the environment due to the following:

- Data from monitoring studies show an absence of MEK in virtually all aquatic and sediment environments sampled, even in areas where it is manufactured, transported, and used;
- MEK volatilizes rapidly from terrestrial and aquatic ecosystems;
- MEK degrades rapidly through biological processes and has a short half-life in air;
- MEK has a low potential to bioaccumulate as a result of rapid loss from the environment (which is further supported by calculated data); and
- MEK has been shown to have very low acute aquatic toxicity, and would not be expected to produce chronic toxicity to aquatic organisms because it rapidly degrades and has limited persistence in aqueous habitats.

IV. DATA ON EMISSIONS AND EXPOSURE

A. Emissions Data

Table 1 below summarizes reported emissions of MEK based on data from the 1994 Toxics Release Inventory (TRI), and indicates the number of total TRI reporting facilities with MEK emissions in different reporting ranges.

TABLE 1

MEK Air Emissions				
No. of Facilities	*Reporting Range lbs/yr*			*% Distribution*
1,587	0	-	20,000	66.4
493	20,001	-	50,000	20.6
160	50,001	-	100,000	6.7
90	100,001	-	200,000	3.8
19	200,001	-	300,000	0.8
16	300,001	-	400,000	0.7
15	400,001	-	700,000	0.6
9	above		700,000	0.4
2,389				

Reference: 1994 TRI Data.

As discussed above in Section I.B, MEK is widely used in many types of solvent-based systems because of its effectiveness. Table 1 shows that, although MEK is used at a large number of facilities, the vast majority of them have very low emissions of MEK. Almost 70 percent of the facilities reporting MEK emissions emitted less than 10 tons in 1994. Over 85 percent of them reported 1994 MEK emissions of less than 25 tons.

Because MEK is manufactured in an enclosed process, emissions from production facilities are low. TRI data for all production facilities, including releases to water and land as well as air emissions, are summarized in Table 2.

TABLE 2

MEK Releases -- Producers (lbs/year)			
Facility	***Air***	***Water***	***Land***
Hoechst Celanese (Pampa, Texas)	75,000	0	0
Exxon Chemical (Baton Rouge, Louisiana)	54,100	420	0
Shell Chemical (Norco, Louisiana)	144,000	0	0

Reference: 1994 TRI Data.

B. Ambient Monitoring Data

MEK has been reported in ambient air at very low concentrations at a limited number of sites in rural and urban locations. Data on ambient air levels of MEK are presented in Appendices F-H. The following is a brief overview of the information presented in those appendices, as well as additional information found in the published literature.

Appendix F includes a table taken from a study conducted by the State of New Jersey Department of Environmental Protection in 1978 (Bozzelli et al. 1980). The table shows that MEK was not found (level of detection was 0.01 ppb) in 170 samples taken at 6 sites.

Thirty-six samples were taken in Elizabeth, New Jersey, near the Exxon Bayway refinery, which at the time was the largest MEK producer in the United States.[14]

An ambient air monitoring study that included MEK has been conducted in the industrial (ship channel) area of Houston since January 1987. The results from the seven monitoring stations in the ship channel during the period of January 1, 1987 to December 31, 1995 show 24-hour average airborne concentrations of MEK (listed in the study as 2-Butanone) from below the level of detection to a high of 31.59 ppb. The mean (long-term average) airborne concentrations of MEK at the seven monitoring sites during the same eight-year period ranged from 0.31 ppb to 0.62 ppb. See Houston Regional Monitoring Report, included in Appendix G.

MEK also was included in a volatile organic screening study conducted in Baton Rouge, Louisiana. The study consisted of three days of sampling (March 6 - 8, 1988) at four locations in the vicinity of the North Baton Rouge Industrial Complex. The four sites were representative of the following settings: (1) rural non-industrial; (2) urban residential near major transportation arteries; (3) urban industrial; and (4) rural industrial. MEK (listed as 2-Butanone) was detected in only one-third of the samples and then only at very low levels. The mean airborne concentration levels for the four sampling locations were 0.3, 1.0, 2.1 and 3.2 ppb, respectively. Additional details are provided in the Final Report, included in Appendix H.

MEK also was detected in air samples taken in Tucson, Arizona in 1982 at an average concentration of 2.8 ppb. In the mountains of Arizona, it was detected at 0.50 ppb (Snider and Dawson 1985). MEK was detected in 70 samples of Los Angeles air in 1980 at

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[14] The Exxon Bayway refinery accounted for 40 percent of domestic production of MEK in 1974-1975. See Lande et al. 1976.

concentrations ranging from 0 to 14 ppb (Grosjean 1982). MEK has been detected in gasoline exhaust at concentrations ranging from less than 0.1 ppm to 2.6 ppm (Verschueren 1983).

C. Air Dispersion Modeling Data for Industrial Facilities

Over the last year, the Ketones Panel has undertaken a program to gather data on the maximum airborne concentrations of MEK to which the public may be exposed. As part of this program, the Panel funded a study by ENSR Corporation to model the maximum off-site concentrations of MEK at a wide variety of facilities emitting MEK, including the largest sources of MEK emissions in the country. The findings of the ENSR study, along with a detailed description of the methodology employed by ENSR, are contained in the report attached at Appendix I (hereinafter referred to as the ENSR Report). This study shows that, even at the largest industrial emitters of MEK, maximum airborne concentrations beyond facility boundaries are well below levels of concern and do not pose a risk to human health or the environment.

The ENSR study was divided into three parts. First, because airborne concentrations are likely to be highest around facilities with the highest emission rates, ENSR individually evaluated maximum off-site concentrations of MEK around each of the 26 facilities reporting MEK emissions of 400,000 pounds or more in 1994. Second, ENSR used a generic model to make conservative estimates of maximum off-site concentrations around smaller facilities that emit MEK. Third, ENSR analyzed the possibility that groups of facilities located in the same area might collectively cause airborne levels of concern. The three parts of ENSR's analysis are discussed below.

1. Air Dispersion Modeling of the Highest Emitters

As the starting point for its modeling program, the Ketones Panel sought to model the maximum off-site concentrations for all facilities reporting MEK emissions greater than 200 tons per year. The Panel selected this threshold based on the methodology that EPA developed to set de minimis values for hazardous air pollutants under Section 112(g) of the Act. See Documentation for De Minimis Emission Rates for Proposed 40 CFR part 63, subpart B (EPA-453/R-93-035). Under the proposed Section 112(g) rule, the de minimis value for a chemical represented the amount that an EPA model facility could emit without posing more than a "trivial" risk to human health or the environment. (The de minimis value for MEK is discussed further in Section V.B of this Petition.) In the rule, EPA proposed to "cap" de minimis levels at 10 tons per year (tpy), but at the same time recognized that, for low toxicity chemicals, emissions of more than 10 tpy would still pose only a trivial risk. 59 Fed. Reg. 15,504, 15,527 (April 1, 1994). Significantly, EPA's methodology may also be used to calculate true "uncapped" de minimis values for different compounds. The uncapped value for MEK would be 2,000 tons (or 4,000,000 pounds) per year.[15] In order to establish a meaningful cutoff point for its modeling exercise, the Panel decided that it would seek to model all facilities with reported emissions that were more than 10 percent of this amount. Thus, it sought to model the maximum off-site concentrations for all facilities reporting MEK emissions of 200 tons (400,000 pounds) or more per year.

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[15] This value was calculated using the outdated RfC of 1.0 mg/m^3. Using the updated RfC of 3.3 mg/m^3, the uncapped de minimis value would actually be 6,600 tpy.

Based on 1994 TRI data, the Panel identified 27 such facilities (including one facility just below the 200 ton cutoff and two facilities at the 200 ton cutoff).[16] The Panel then worked with ENSR to develop a detailed questionnaire to gather the information that would be necessary to model the maximum off-site concentrations at these facilities. This questionnaire was sent to each of the identified facilities, and representatives from the Panel also contacted each of the facilities to encourage their participation. The necessary information (including preexisting modeling results for 2 facilities) was received from 16 of the 26 facilities, including the 4 highest emitters and 7 of the top 10.

In order to conduct modeling for the other 11 facilities identified as top emitters, the Panel and ENSR attempted to obtain the necessary site-specific information from public sources. For each facility, the Panel conducted a search on EPA's Aerometric Information Retrieval System (AIRS) database. The Panel also contacted state and local regulatory officials to determine whether a Title V permit application or similar document had been submitted for any of the remaining facilities. Title V permit applications were obtained for 4 facilities. Based on the information available from the AIRS database and permit applications, ENSR was able to conduct refined dispersion modeling for 5 of the 11 facilities that did not provide information

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[16] The 1994 TRI data indicate that Sun Refining & Marketing Co. was one of the 26 facilities that reported MEK emissions of 400,000 pounds or more in 1994. This facility has informed the Panel that it recently conducted emissions tests which indicate that its 1994 MEK emissions to the air were overstated by a significant amount, and actually were well below the cut off level of 400,000 pounds. The 1995 Form R for this facility more accurately reports air emissions of 241,000 pounds. Based on this information, the Panel has not included dispersion modeling results for this facility in Tables 3 and 4.

directly to ENSR. Thus, the data necessary for site-specific modeling was obtained -- either from the facilities or from public sources -- for 21 of the 26 highest emitters, including the 6 facilities with the highest emissions and 13 of the top 15.[17]

Using this data, ENSR performed an air dispersion modeling analysis for each facility using EPA's "Tiered Modeling Approach for Assessing Risks Due to Sources of Hazardous Air Pollutants" (1992). This approach uses three successively rigorous modeling techniques. Tier 1 requires only limited source information and an EPA look-up table, and provides the most conservative predictions of maximum concentrations. Tier 2 requires additional source information and an EPA screening level computer program, and generates predictions that are somewhat more realistic than Tier 1 predictions. Tier 3, which requires extensive data from the source and uses EPA's most advanced dispersion modeling techniques, provides the most realistic predicted maximum concentrations. Because each tier provides a less conservative (and more realistic) prediction, Tier 3 modeling was not performed for a facility if Tier 2 modeling predicted maximum concentrations below the relevant health benchmark (3.3 mg/m^3 and 33 mg/m^3 for annual and 24-hour concentrations, respectively).

The results of the ENSR modeling study of the highest emitters are shown on Tables 3 and 4. Table 3 shows maximum annual off-site concentrations; Table 4 shows

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17 There were 6 facilities for which no site-specific data could be obtained. As discussed in the ENSR Report, ENSR estimated maximum annual off-site concentrations at these 6 facilities using the generic approach developed to evaluate ambient concentrations around smaller sources. This approach is described in Section IV.C.2 below. Maximum predicted annual off-site concentrations at each of these 6 facilities was less than 1.0 mg/m^3. ENSR Report at pp. 2-3, 4-3 (Table 4-2), 5-1.

maximum 24-hour off-site concentrations. In both tables, facilities are listed according to Tier 2 modeling results in descending order.

TABLE 3

Air Dispersion Modeling Results for Highest-Emitting MEK Sources			
Maximum Annual MEK Concentrations (mg/m^3) (RfC = 3.3 mg/m^3)			
Site†	Tier 1	Tier 2	Tier 3
E17	29.0	12.33	1.18
E9	33.0	11.2	0.91
E28	13.7	4.75	0.81
E7	14.5	3.97	0.50
E25	38.1	3.37	0.42
E4	17.1	2.99	0.55
E3	12.3	2.65	0.63
E20	19.4	1.77	-
E33*	5.5	1.56	-
E19*	8.5	1.42	-
E21	14.6	1.37	-
E14*	6.9	1.23	-
E29	26.5	1.13	-
E16*	6.9	0.69	-
E23	9.9	0.46	-
E8	9.7	0.42	-
E32	6.2	0.18	-
E1	9.2	0.12	-
E11*	0.2	0.1	-
E15	-	-	0.03**

* Model input parameters based on site-specific data available from public sources rather than data provided by the facility itself.

** Based on dispersion modeling results provided by individual facility.

† Companies submitted the information necessary to conduct the modeling under conditions of confidentiality. For this reason, facilities are not identified by name and modeling results cannot be presented side-by-side with emissions data.

As shown on Table 3, maximum off-site annual concentrations of MEK for the highest-emitting facilities are all below the updated RfC of 3.3 mg/m^3. Indeed, for most facilities, it was not necessary to conduct Tier 3 modeling, as the Tier 2 results for two-thirds of the facilities showed predicted maximum annual concentrations below the RfC. Only one facility (E17) had a Tier 3 maximum off-site concentration above 1.0 mg/m^3. In this case, however, the highest predicted concentration (1.2 mg/m^3) was found at the vehicular entrance to an adjacent industrial facility where there are no environmental or human receptors. The second-highest predicted off-site concentration at this facility was 0.81 mg/m^3. Maximum annual Tier 3 concentrations at all other facilities were also below 1.0 mg/m^3.

TABLE 4

Air Dispersion Modeling Results for Highest-Emitting MEK Sources			
Maximum 24-hour MEK Concentrations (mg/m^3) (Benchmark = 33.0 mg/m^3)†			
Site††	Tier 1	Tier 2	Tier 3
E17	186.8	86.3	12.82
E9	196.2	64.0	7.42
E28	137.0	33.3	5.24
E7	81.9	27.8	4.67
E3	58.2	18.5	2.44
E25	232.3	16.85	2.77
E4	73.0	15.63	7.57
E20	108.5	12.39	-
E19*	47.6	9.91	-
E21	146.3	9.57	-
E14*	38.3	8.58	-
E33*	35.0	8.27	-
E29	144.2	7.91	-
E16*	38.5	4.86	-
E23	115.2	3.21	-
E8	54.4	2.93	-
E32	35.0	1.23	-
E11*	1.2	0.72	-
E1	36.7	0.59	-
E15		-	0.15**

* Model input parameters based on site-specific data available from public sources rather than data provided by the facility itself.

** Based on dispersion modeling results provided by individual facility.

† This health benchmark is based on the updated RfC of 3.3 mg/m^3, modified only to eliminate the uncertainty factor of 10 for extrapolating from subchronic to chronic exposures.

†† Companies submitted the information necessary to conduct the modeling under conditions of confidentiality. For this reason, facilities are not identified by name and modeling results cannot be presented side-by-side with emissions data.

Maximum 24-hour off-site MEK concentrations were compared to a health benchmark of 33 mg/m^3 -- the updated RfC modified to eliminate the uncertainty factor of 10 that is used to extrapolate from subchronic to chronic exposure. As shown on Table 4, the maximum 24-hour concentrations were all well below this benchmark. The highest predicted 24-hour value was 12.8 mg/m^3 -- at the same location where the highest annual concentration was identified. As noted above, there are no human or environmental receptors at this location. The second-highest 24-hour off-site concentration at this facility was 8.3 mg/m^3. In all other cases, the maximum Tier 3 predicted 24-hour concentrations were also below 10 mg/m^3. The 24-hour values are based on the worst-case conditions occurring in any 24-hour period over the last 5 years, and are therefore very conservative.

It is important to recognize that the methodology used by ENSR was not intended to represent actual population exposure. Rather, it was designed to identify the highest annual and 24-hour off-site concentrations that might occur around each facility. In all cases, the modeled maximum concentrations are near facility boundaries, and it is unlikely that there is continuous exposure at any of these locations. Further, because the methodology is designed to predict **maximum** off-site concentrations, it incorporates a number of conservative assumptions. Actual average concentrations are likely to be lower, and could be lower by an order of magnitude or more. Thus, the results of ENSR's air dispersion modeling analyses likely overpredict actual exposures.

2. Air Dispersion Modeling of Smaller Sources

The Panel also recognized that there could be relatively high off-site exposures around smaller MEK sources due to such things as unusual dispersion climatology, lower

emission release heights, and proximity of nearby residents. Therefore, ENSR developed an approach for analyzing maximum off-site concentrations around smaller facilities that emit MEK. All facilities that reported MEK emissions of 10 tons or more on the 1994 TRI were divided into source categories based on their two-digit SIC codes. As discussed below, ENSR developed model facilities for each source category in which no facility was individually modeled under the first part of its analysis. It then used a generic EPA model to predict maximum off-site concentrations for facilities in each source category.

As described more fully in the ENSR Report, the air dispersion model used to evaluate potential exposures around smaller sources was based on the model developed by EPA as part of the Agency's rulemaking under Section 112(g) of the Clean Air Act. This is a conservative model that allows the prediction of maximum annual exposures based on two parameters: emissions release height and distance to the nearest receptor. The EPA model incorporates the following conservative assumptions:

- emissions all emanate from a single point;
- emissions have negligible exit velocity (10 cm/second);
- emissions are released at ambient temperature; and
- emissions are subject to worst-case aerodynamic building downwash.

For purposes of the Section 112(g) rulemaking, the Agency applied the model based on median dispersion climatology developed from 314 weather stations located throughout the United States. To predict maximum annual concentrations around smaller sources of MEK, ENSR adjusted the model to incorporate worst-case dispersion climatology.

ENSR then assigned each of the source categories identified with MEK sources to one of the following "dispersion categories" based on the assumptions set forth below:

- Heavy: Major facilities located in industrial areas on relatively large sites. (Stack height = 15 meters; distance to receptor = 200 meters)
- Medium: Moderate size facilities located in light industrial or commercial areas on smaller sites. (Stack height = 10 meters; distance to receptor = 150 meters)
- Light: Smaller facilities located on relatively small sites in mixed-use areas, where emissions are likely to be released from roof vents in one-story buildings. (Stack height = 5 meters; distance to receptor = 100 meters)

The model was then used to predict maximum annual concentrations for facilities representing each source category in which at least one facility reported more than 10 tons of MEK emissions in 1994. The predicted maximum concentrations for each source category are based on the emission rates reported by the facilities reporting the highest and second highest emissions in each category. The results of this analysis are presented in Table 5.

TABLE 5

Air Dispersion Modeling Results for Smaller MEK Sources						
Maximum Annual MEK Concentrations (mg/m^3) (RfC= 3.3 mg/m^3)						
SIC Code	**Source Category Description**	**Dispersion Category**	**Highest Emission Rate in SIC Code (lbs)**	**Predicted Concentration from Highest Rate (mg/m^3)**	**2nd Highest Emission Rate in SIC Code (lbs)**	**Predicted Concentration from 2nd Highest Rate (mg/m^3)**
23	Apparel	Light	179,264	0.735	74,622	0.0306
24	Lumber	Medium	378,865	0.345	172,000	0.157
25	Furniture	Light	195,801	0.562	136,004	0.558
26	Paper	Medium	362,560	0.330	248,158	0.226
27	Printing	Medium	185,000	0.168	176,000	0.160
28	Chemical	Heavy	282,754	0.110	248,158	0.097
31	Leather	Light	112,211	0.198	42,900	0.176
32	Concrete	Light	127,000	0.521	120,007	0.492
45	Air Transportation	Medium	24,000	0.022	NA	NA
51	Wholesale Trade	Light	152,800	0.626	113,316	0.465
87	Engineering	Light	24,930	0.102	NA	NA
97	National Security	Heavy	185,757	0.072	59,130	0.023

Based on this analysis, ENSR concluded that "[g]eneric dispersion modeling of lesser MEK emitters indicates that maximum annual off-site concentrations are likely to be below 1 mg/m^3 in all cases, and well below 1 mg/m^3 in most cases." ENSR Report at p. 1-2.

Thus, based on an assessment of a wide range of sources and source categories, including both large and small sources, it appears that maximum off-site concentrations of MEK are well below levels of concern.

3. Potential Impacts from Groups of Sources

Based on its dispersion modeling study, ENSR concluded that it was "highly improbable" that groups of sources collectively could cause airborne MEK concentrations of concern. ENSR Report at p. 6-1. It noted that, because maximum off-site concentrations at major sources were consistently below 1 mg/m^3, it would be virtually impossible for emissions from a group of sources to cause ambient concentrations in excess of the RfC unless three or more major sources were virtually co-located. In this regard, ENSR pointed out that no two facilities on the list of the highest MEK emitters were located in the same geographic area.

To confirm this conclusion, the Panel analyzed the TRI data on MEK emissions to evaluate the possibility that there might be groups of facilities that collectively cause significant airborne concentrations of MEK, even though no single facility would be responsible for off-site concentrations in excess of the RfC. As the starting point for this analysis, the Panel identified every facility in the country that reported MEK emissions of more than 10 tons in 1994, including both point and fugitive emissions. For the year 1994, there were exactly 800 such facilities. Of this number, 709 facilities were located in postal zip codes in which there was no other facility reporting MEK emissions greater than 10 tons. Analysis of the other 91 facilities showed that there is no significant grouping of sources that emit MEK. A list of these facilities (grouped by ZIP code) and their 1994 MEK emissions is attached as Appendix J.

As shown in Appendix J, the 91 facilities are distributed fairly evenly over 37 different ZIP codes. Only one ZIP code contained as many as 5 facilities, and the combined emissions from these 5 facilities were only about 100 tons in 1994. Only three ZIP codes contained groups of facilities that collectively emitted more than 200 tons in 1994. One of these areas included a facility (Resilite Sports) that was modeled as one of the top 26 MEK emitters, and the modeling results showed that off-site concentrations from that facility were well below levels of concern. The only other facility in the same ZIP code emitted less than 15 tons in 1994 -- an amount that would not contribute significantly to ambient concentrations in the area even if it were co-located with the Resilite Sports facility.

The other two ZIP codes with total emissions in excess of 200 tons each contained two principle sources. ENSR did not individually model these two pairs of facilities, but used the modeling results from other higher-emitting sources to evaluate the potential for these facilities to have significant overlapping impacts. ENSR's modeling study of the highest-emitting MEK sources showed that airborne concentrations fall off rapidly with distance beyond facility boundaries. For each of the 7 facilities with the highest predicted off-site MEK concentrations, maximum predicted annual concentrations were below 0.5 mg/m^3 within 175 meters from the facility fenceline. Therefore, ENSR conservatively estimated that two facilities would not cause significant overlapping impacts unless they were located within 350 meters of each other. For the two pairs of facilities located in the same ZIP codes, longitude and latitude data from the TRI database showed that the distance between the two facilities was significantly greater than 350 meters. Based on its modeling study and its review of the ZIP code data, ENSR concluded that "combined impacts from multiple sources of MEK will not result in ambient

levels greater than either 1.0 mg/m^3 (annual average) or 10 mg/m^3 (24-hour average)." ENSR Report at p. 6-2.

D. Effect of Delisting on Emissions and Ambient Concentrations

Because of MEK's status as a HAP, companies currently are discouraged from using it. In some cases, product formulators and users must comply with regulatory limits on the HAP content of their products. In other cases, companies are likely to reduce their use of HAPs in order to avoid the need to install maximum available control technology (MACT) under section 112(d) of the Act. Even in the absence of regulatory requirements, companies often try to avoid the use of chemicals that are labeled as HAPs. Therefore, if MEK is removed from the HAP list, usage of MEK is likely to increase.

For several reasons, however, MEK emissions are unlikely to increase substantially. First, MEK is used primarily in paints and coatings and, to a lesser extent, in inks and adhesives. In these and other solvent applications, MEK is rarely used by itself. Typically, MEK is part of a solvent blend that must be carefully formulated to achieve the proper performance characteristics, including such things as evaporation rate, surface tension, solvent balance, and flash point. Although there is flexibility to increase the use of MEK in many solvent blends, there are inherent limits on the amount of MEK (or any other single solvent) that can be used in any formulation.

Second, because MEK is used primarily in solvent blends, there will often be other HAPs that are used in the same application. In many cases, the facilities where such applications are used will be required to meet "maximum available control technology" (MACT) standards and will need to install control technology to reduce their HAP emissions. In most

cases, such technology will reduce all solvent emissions, including emissions of MEK. Therefore, although MEK would no longer be listed as a HAP, the implementation of MACT standards will control MEK emissions along with emissions of other chemicals.

Third, MEK will continue to be regulated as a VOC. Many areas of the country have not yet reached attainment with the national air quality standard (NAAQS) for ozone and must reduce VOC emissions in order to meet the NAAQS standard. Emissions of solvents, including MEK, are subject to increasingly stringent standards under both federal and state programs designed to control ozone formation. Therefore, emissions of MEK will continue to be regulated even after it is removed from the HAP list.

Significantly, the monitoring and modeling data discussed above show that, even if MEK emissions were to increase significantly, ambient concentrations would be expected to remain well below levels of concern. Actual monitoring data in a number of areas, including industrial areas, indicate that ambient concentrations of MEK are very low. The air dispersion modeling study conducted by ENSR showed that, in most cases, maximum annual off-site concentrations around facilities emitting MEK should be below the RfC by an order of magnitude or more. Even at the worst-case off-site locations around the largest sources of MEK, ambient levels are below the RfC by a factor of 3 or more. Therefore, even significant increases in MEK emissions would not pose an appreciable risk to human health or the environment.

Finally -- and perhaps most importantly -- any increase in MEK emissions is likely to be more than offset by decreases in emissions of other solvents. As discussed in the next section of this Petition, MEK is a highly effective solvent. In many applications, relatively small amounts of MEK may be used to perform the same function served by other, less efficient

solvents. For this reason, it is widely used in high-solids coatings, which offer the only feasible approach for reducing VOC emissions from certain types of coating operations. Because of MEK's efficiency as a solvent, increases in MEK emissions will in many cases represent an overall decrease in VOC emissions.

V. OTHER REASONS FOR DELISTING MEK

As discussed above, MEK meets the statutory delisting criteria and should therefore be removed from the HAP list. EPA should be aware, however, that there are additional considerations that weigh in favor of removing MEK from the Section 313 list. These additional considerations are discussed below.

A. Delisting MEK Will Help to Reduce VOC Emissions from Many Coating Operations

As noted above, MEK is especially valuable in the formulation of high solids coatings, which are increasingly used to reduce VOC emissions from industrial and commercial coating operations. MEK is widely recognized for having "outstanding solvent properties" for a wide variety of resins. See Agency for Toxic Substances and Disease Registry, Toxicological Profile for 2-Butanone (July 1992) at p. 59. Compared to the available non-ketone alternatives, a smaller amount of MEK may be used to perform the same function. The use of MEK therefore allows the formulation of coatings with higher solids content and lower VOC emissions.

Over the last decade, EPA and many state agencies have sought to reduce VOC emissions from coating operations and other commercial applications that involve the use of organic solvents. In some cases -- particularly those involving large-scale coating operations -- the most effective approach for reducing VOC emissions is to install a solvent recovery system

or other type of control device. In other cases, companies have reduced their VOC emissions by switching from solvent-based technologies to alternative, non-solvent technologies. A number of coating operations, for example, have switched from conventional solvent-based coatings to waterborne or powder coatings.

In many cases, however, these options are simply not feasible. For example, in many wood coating applications, water-based finishes cannot be used because they are absorbed into the substrate and raise the grain of the wood. Although a control device may be technically feasible for some wood finishing operations, EPA has determined that many such operations are simply too small to justify the installation of a control device. Where it is not practical to use a control device or a non-solvent technology, EPA has recognized that the best alternative is to use products that can accomplish a given task with the least possible amount of solvent. For coating applications, this generally means a switch from conventional coatings to "high-solids" coatings. In several recent rulemakings, EPA has adopted standards that will effectively require the use of such coatings in certain industries. See, e.g., 61 Fed. Reg. 19005 (April 30, 1996) (proposed rule; automobile refinishing coatings); 60 Fed. Reg. 62930 (Dec. 7, 1995) (final rule; wood furniture coating operations); 60 Fed. Reg. 64330 (Dec. 15, 1995) (final rule; shipbuilding coating operations).

The amount of solids in a coating is limited by the ability of the solvent to dissolve the resins and retain them in solution until the coating is applied. After the coating is applied, the solvent evaporates into the air, leaving behind a hard, uniform finish. Thus, the more effective the solvent, the higher the proportion of solids and the lower the emissions into the air.

EPA recognized this fact in its recent rule to reduce emissions from shipbuilding operations. See 60 Fed. Reg. at 64330. In this rulemaking, EPA acknowledged that the use of highly efficient solvents such as MEK is the preferred environmental alternative in many coating applications, even though such solvents may be listed as HAPs. Although the primary purpose of the rule is to control HAP emissions, EPA designed the rule to minimize VOC emissions as well. Thus, the Agency adopted regulatory standards that effectively require the use of higher-solids coatings in the shipbuilding industry.

MEK and methyl isobutyl ketone (MIBK) – another ketone solvent with similar solvency – are two of the most efficient solvents available to coating formulators. Because they are currently listed as HAPs, a formulator may need to increase the HAP content of a coating in order to reduce its VOC content. The Agency explicitly recognized this tradeoff in the preamble to the proposed rule. It noted that a coating reformulated to reduce its HAP content may have "higher VOC content than the one it replaces," and went on to say that "the HAP to VOC ratio may even increase when a company develops a new reformulation with lower VOC." 59 Fed. Reg. at 62688. The Agency also noted, however, that even where the HAP to VOC ratio in the coating increases, "the absolute HAP emissions are likely to go down," presumably because higher solids coatings allow more coverage per gallon of coating. Id. (emphasis in original).

The Agency addressed this issue by setting identical limits for the VOC and HAP content of the coatings covered by the shipbuilding rule. The rule sets a limit on the amount of "volatile organic hazardous air pollutants" (VOHAPs) that can be used in specified types of coatings. Because VOHAPs are defined to include both HAPs and VOCs, a formulator may use any solvent up to the VOHAP content limit for each coating. This approach encourages the use

of higher solids coatings and eliminates the incentive for formulators to use less efficient solvents that must be used in greater volumes. Thus, the rule recognizes that highly efficient solvents such as MEK are the preferred environmental approach for reducing overall emissions from many coating applications.

Because MEK is currently listed as a HAP, however, companies are discouraged or even prevented from using it -- even where it would allow them to reduce their VOC emissions by switching to higher-solids coatings. In some industries, such as the wood furniture industry, facilities must comply with regulatory limits on the HAP content of their coatings. In other industries, as already stated, companies are likely to reduce their use of HAPs in order to avoid the need to install maximum available control technology (MACT) under section 112(d) of the Act. Even in the absence of regulatory requirements, companies often try to avoid the use of chemicals that are labeled as HAPs. The simple fact that MEK is listed as a HAP discourages companies from using it -- even in applications where it would provide a clear environmental benefit. Removing MEK from the HAP list would eliminate this disincentive and benefit the environment by facilitating the use of higher-solids coatings.

B. EPA Has Recognized in Other Contexts that MEK Has Relatively Low Toxicity

In two recent rulemakings, EPA has specifically evaluated the health effects of exposure to MEK. In both cases, EPA's analysis confirmed that MEK has relatively low toxicity.

1. Proposed Rule Under Section 112(g) of the Clean Air Act

Under Section 112(g) of the Clean Air Act, EPA is required to evaluate the relative risks of the 189 chemicals and chemical categories that are listed as HAPs under section

112(b) of the Act. On April 1, 1994, the Agency published a proposed rule under section 112(g) that included a detailed system for ranking and setting "de minimis values" for the various listed chemicals, including MEK. 59 Fed. Reg. 15,504.

The proposed rule defined the de minimis value as the amount of a chemical that, based on an EPA model, a typical facility could emit without posing more than a "trivial" risk to human health or the environment. For compounds such as MEK that are non-carcinogens, the values were designed to ensure that public health was protected with an "ample margin of safety." Id. at 15, 525. The de minimis values in the proposed rule range from 0.0000006 tons per year to 10 tons per year. For policy reasons unrelated to risk, EPA "capped" de minimis levels at 10 tons per year, but at the same time recognized that, for several low toxicity chemicals, emissions of more than 10 tons a year would still pose only a trivial risk. Id. at 15,527. Not surprisingly, the proposed de minimis level for MEK was set at the 10 ton cap.

Significantly, however, EPA's methodology may be used to calculate the true "uncapped" de minimis value for MEK. This approach is still conservative for at least three reasons. First, as noted above, EPA's approach for setting de minimis values was specifically designed to allow an "ample margin of safety." Second, it is based on the RfC for MEK in the IRIS database, which was not derived using EPA's current guidelines for setting RfCs. As discussed above at pp. 24-25, if EPA's current RfC guidelines are followed, the RfC for MEK is higher by a factor of more than three. And third, although the EPA model used to calculate the de minimis values is not a "worst-case" model, the Agency has recognized that it does incorporate a number of conservative assumptions. Id. at 15,526. Nevertheless, based on this methodology, the uncapped de minimis level for MEK would be 2000 tons per year if calculated

using an RfC of 1.0 mg/m^3. Based on the most recent TRI data, the facility with the highest MEK emissions in the country emits barely half of this amount, and only 5 facilities in the country reported MEK emissions that are even 25 percent of this amount. If the updated RfC of 3.3 mg/m^3 is used in EPA's model, then the uncapped de minimis value would actually be 6,600 tons per year.

In the proposed section 112(g) rule, EPA also developed a system for ranking the relative hazards of the listed chemicals. Under this system, EPA put together a list of "threshold pollutants" that were not considered to be "high concern" pollutants, and were believed to pose the least risk of any of the pollutants on the list. Not surprisingly, MEK was listed as a threshold pollutant. For ranking the relative hazards of the compounds on the threshold list, the Agency assigned a "composite score" for each chemical based on the severity of any health effect caused by the chemical in test animals and the dose at which the effect is likely to occur. Under this system, a chemical could receive a composite score from 1 - 100, although the pollutants on the threshold list were all assigned scores between 2 and 46.

In the proposed section 112(g) rule, the Agency has assigned a composite score of 10 to MEK, indicating that it is among the least hazardous of the chemicals on the list (approximately 177 out of 189). The composite score of 10, however, was based on a 1955 single dose study by LaBelle and Brieger, which EPA has rejected as a basis for setting exposure standards. If the composite score for MEK were based on the current data in EPA's IRIS database, it would be even lower than 10.

2. Final SNAP Rule

Under section 612 of the Clean Air Act, EPA has developed a program -- called the Significant New Alternatives Policy (SNAP) program -- to identify acceptable substitutes for chemicals that are being phased out of production because they deplete the stratospheric ozone layer. 59 Fed. Reg. 13,044 (March 18, 1994). Under the SNAP program, the Agency specifically evaluated the toxicity of MEK and listed it as an acceptable substitute in a number of applications. In the final SNAP rule, EPA discussed concerns about possible risks posed by petroleum hydrocarbons and concluded that these risks were relatively small and were adequately addressed by existing regulations and work practices. The Agency then discussed the use of oxygenated hydrocarbons and stated that "two of the typical oxygenated hydrocarbons examined in the Agency's risk screen, methyl ethyl ketone and methyl isobutyl ketone, also have comparatively low toxicity." 59 Fed. Reg. at 13,120. Thus, EPA has recognized that MEK has relatively low toxicity and that the use of MEK as a substitute for ozone-depleting chemicals actually helps to protect the environment.

C. The Inclusion of MEK on the Initial HAP List Was Not Based on a Finding of Adverse Health or Environmental Effects

The inclusion of MEK on the initial HAP list was not based on a finding of adverse health or environmental effects. The initial HAP list was developed from the list of chemicals that are reportable under Section 313 of EPCRA. The Section 313 list was a compendium of the New Jersey "Environmental Hazardous Substance List" and a Maryland "Survey List." These two lists were combined in Committee Print No. 99-169 of the Senate Committee on Environment and Public Works, entitled "Toxic Chemicals Subject to Section 313 Of The Emergency Planning and Community Right-To-Know-Act of 1986." The combined list

constitutes the initial list of chemicals subject to the reporting requirements of Section 313. See Section 313(c).

At the time EPCRA was enacted, MEK was not on the New Jersey list. When New Jersey enacted the Worker and Community Right-to-Know Act (codified at N.J.A.C. § 7:1G-1, et seq.), it compiled a list of 250 chemicals for careful review based on three criteria: whether the chemical (1) presented a public health hazard; (2) was an environmental contaminant; or (3) was present in the State in quantities of 10,000 pounds or more. New Jersey then gathered information about the production, use and effects of these 250 chemicals from a number of sources and evaluated each chemical for inclusion on its list based on two criteria: (1) evidence of significant production in New Jersey and (2) evidence of health or environment effects such as carcinogenicity, teratogenicity, mutagenicity, acute toxicity, persistence and ability to bioaccumulate. See New Jersey Department of Environmental Protection, "Worker and Community Right-to-Know Basis and Background Document." Notably, New Jersey did not include MEK in its final Hazardous Substance list.

MEK was included in the Maryland Survey List, but its inclusion did not reflect a finding of adverse health or environmental effects. The Maryland Survey List was informally developed, based on a variety of federal and state lists, for purposes of information gathering by the State regarding chemical usage in Maryland. The Survey List was eventually used to survey Maryland businesses to determine the production and use levels in the State of Maryland for each chemical.

Therefore, the inclusion of MEK on the initial HAP list does not reflect a determination by Congress, EPA or anyone else that MEK meets the listing criteria. Moreover,

the circumstances surrounding the creation of the initial list do not create any presumption against delisting.

CONCLUSION

The Ketones Panel respectfully submits that MEK meets the delisting criteria set forth in Section 112(b)(3)(C) of the Clean Air Act. Accordingly, the Administrator should grant this Petition and remove MEK from the list of chemicals that are regulated as hazardous air pollutants under the Clean Air Act.